Observational Study

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Gensini score values for predicting periprocedural myocardial infarction

An observational study analysis

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Abstract

The Gensini score (GS) is a convenient, powerful tool for assessing the severity and complexity of coronary artery diseases. Our research investigated the relationship between the GS and periprocedural myocardial infarction (PMI).

We recruited 4949 patients (3366 men, 1583 women; mean age 66.45 ± 10.09 years) with a single coronary artery revascularization. Based on the tertile of the GS 20 and 36, the population was divided into 3 groups: Low Group ($0 < GS \leq 20$, N = 1809); Intermediate Group ($20 < GS \leq 36$, N = 1579); High Group ($GS > 36$, N = 1561). PMI3 represented the endpoint for cTnI > 3-fold upper reference limit, while PMI5 represented the endpoint for cTnI > 5-fold upper reference limit.

The incidence of PMI of High Group was statistically higher than that of Intermediate Group ($P < .05$), while that of Intermediate Group was statistically higher than Low Group ($P < .05$). With the adjustment of some general variables, GS was an independent significantly predictor for PMI3 ($\beta = 0.006$, $P < .05$) and PMI5 ($\beta = 0.007$, $P < .05$). Following receiver operating characteristic curve analysis, the optimal cut-off value to predict PMI are 22.5 for PMI3 and 27 for PMI5.

The GS was an independent predictor of PMI in the single-coronary revascularization population. Additionally, the 22.5 of GS was the optimal cut-off value for determining the presence of PMI3, while the 27 of GS for PMI5.

Abbreviations: ACC = American College of Cardiology, AHA = American Heart Association, CAD = coronary artery diseases, CK-MB = creatine kinase-MB fraction, cTnI = cardiac troponin I, CVD = cardiovascular diseases, GS = Gensini score, PCI = percutaneous coronary intervention, PMI = periprocedural myocardial infarction, ROC = receiver operating characteristic, SCAI = the Society for Cardiovascular Angiography and Interventions, URL = upper reference limit.

Keywords: Gensini score, periprocedural myocardial infarction

1. Introduction

It is well-known that the cardiovascular diseases (CVD) are the leading cause of deaths all over the world. In particular, coronary artery diseases (CAD) are the major cause (43.8%) of deaths attributable to CVD in the United States.[1] Give this, percutaneous coronary intervention (PCI) is becoming the most popular treatment for CAD. While there have been technical advances in the PCI process, there is still a high incident rate of periprocedural myocardial infarction (PMI) rate of approximately 5% to 30% continues to be reported.[2,3] Therefore, it is critical that clinical practitioners discover a unique predictor for the presence of PMI as a significant orientation in the cardiology field.

The Gensini score (GS) is a convenient and powerful tool for assessing the severity and complexity of narrowing in the coronary arteries,[4] as are the SYNTAX score,[5] American Heart Association (AHA)/American College of Cardiology (ACC) classification,[6] and LEAMAN score.[7] In the past few decades, GS has played a key role in the description of CAD degree. In addition, GS may also be used to stratify risk for long-term prognosis.[8] While many authors consider GS a primary outcome for evaluating the severity of CAD before PCI, a few authors have explored the relationship between GS and after-procedural complications, in particular PMI.[9]

PCI resulting in direct instrumentation and manipulation of the coronary arterial vasculature predispose patients to ischemic

YW and QL contributed equally to this work.

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The datasets generated during and/or analyzed during the current study are not publicly available, but are available from the corresponding author on reasonable request.

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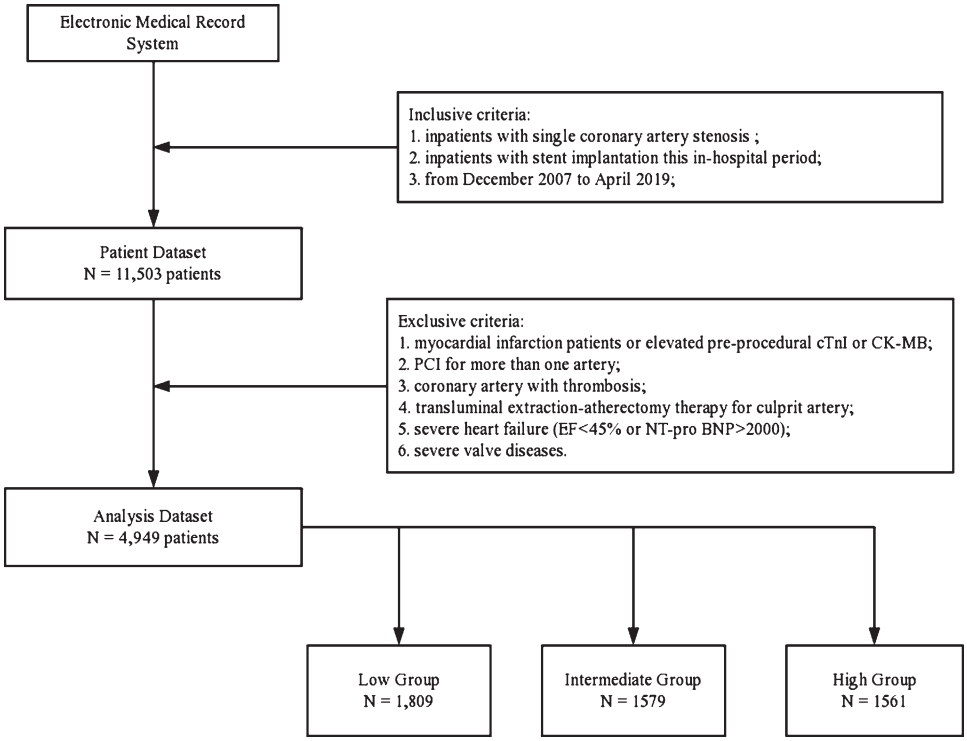

Figure 1. Study design flowchart.

events that can lead to myocardial necrosis.[10] Nowadays, many factors have been approved to be related with the process of PMI, which could be categorized as patient-related factors, lesion-related factors, and procedure-related factors. In totality, these factors identify patients with increasing atherosclerotic disease burden, increased thrombotic risk,[11] and with neurohormonal activation that predisposes to either macrovascular complications (side branch occlusion or macroembolization) or microvascular obstruction (distal embolization of microparticles), unifying the pathophysiologic basis of myocardial necrosis after PCI.[12]

Above all, our hypothesis is that GS is an independent predictor for the presence of PMI in the broad population and we could determine a special cut-off point of GS for the prediction of PMI, according to the definitions of the Society for Cardiovascular Angiography and Interventions (SCAI) and fourth universal definition of myocardial infarction.

2. Methods and Materials

2.1. Study design and patient population

This was a single-center, retrospective research that took place in the Sir Run Run Shaw Hospital (Hangzhou, China) from December 2007 to April 2019. (1) inpatients with single coronary artery stenosis (left main, left anterior descending, left circumflex, or right coronary artery); (2) inpatients who had a stent implanted at the hospital during the time period. Patients were excluded if they had myocardial infarction, elevated pre-procedural cardiac troponin I (cTnI), creatine kinase-MB fraction (CK-MB), PCI for more than 1 artery, a coronary artery with thrombosis, transluminal extraction-atherectomy therapy for culprit artery, severe heart failure (Ejection Fraction < 45% or NT-pro BNP > 2000) or severe valve diseases (Fig. 1). All the study protocols were in accordance with the Declaration of Helsinki, and all patients provided written informed consent. The Institutional Ethics Research Committee of Sir Run Run Shaw Hospital has approved the study (approval ID: 20200803-34), and the approved file was provided in the supplementary field named "Etheical_information.pdf".

2.2. Definitions and assessment of PMI

The primary endpoint was PMI, following the definitions of the SCAI[13] and the Universal Definition of Myocardial Infarction,[14] the Cut-Off value of cTnI for PMI was >3-fold or 5-fold upper reference limit (URL) in addition to symptoms, ECG changes, angiographic findings or new regional wall motion abnormalities 48 hours after the procedure. In our study, PMI3 represented the endpoint for cTnI > 3-fold URL, while PMI5 represented the endpoint for cTnI > 5-fold URL. The URL of cTnI in this study is 0.11 ng/mL.

Cardiac biomarkers and electrocardiograms were systematically assessed for all participants before and after index PCI or staged procedure to identify PMI. cTnI levels were evaluated every 8 hours after the PCI, while 24 to 48 hours dynamic monitoring was carried out after the procedure if necessary.

2.3. Gensini score

GS was initially put forward in 1983,[4] and had a pivotal role in the stratification for determining the severity of CAD. Since 1983, many cardiology centers have proved the validation of this severity score system for CAD. Precisely, the GSs can be calculated using the following steps: (1) define the concentric or eccentric luminal narrowing degrees (stenosis of 1% to 25%, 26% to 50%, 51% to 75%, 76% to 99%, 100% are given a score of 1, 2, 4,8, 16, 32, respectively); (2) each changeable segment of coronary has a specific coefficient based on its importance for blood supply to the heart; (3) the summation of all changeable segment scores (narrowing score * coefficient) is the final GS.

Coronary angiography was performed in our department following the standard procedure.[15,16] All participants were

Table 1

Demographic data of different groups.

Categories	Variables	Low group (N = 1809)	Intermediate group (N = 1579)	High group (N = 1561)	P	P (low vs inter)	P (low vs high)	P (inter vs high)
General information	Gender, male%	64	67	74		.06		
	Age, yr	65.61 a 9.94	66.73 a 9.84	67.14 a 10.42				.26
	BMI, kg/m^2	23.93 a 14.56	23.44 a 7.03	23.71 a 5.61	.58	.33	.43	.86
	SYB, mmHg	133.94 a 39.73	133.39 a 23.07	135.19 a 39.92	.43	.85	.22	.31
	DBP, mmHg	74.58 a 13.41	74.20 a 20.98	73.59 a 13.31	.15	.98	.09	.10
	UAP, yes%	52	54	51	.22	.34	.40	.08
Medical history	Hyper, yes%	65	68	70				.23
	DM, yes%	19	23	28				
	Smoking, yes%	40	42	44	.05	.16		.32
	Drinking, yes%	33	31	31	.23	.17	.12	.86
	F-CVD, yes%	9	9	10	.28	.80	.13	.23
Biochemistry results	TC, mmol/L	4.18 a 1.08	4.26 a 1.16	4.27 a 1.24				.71
	HDL-C, mmol/L	1.09 a 0.30	1.08 a 0.28	1.03 a 0.28		.30		
	LDL-C, mmol/L	2.17 a 0.82	2.28 a 0.88	2.30 a 0.93				.45
	VLDL-C, mmol/L	0.92 a 1.09	0.90 a 1.16	0.99 a 1.29	.10	.61	.10	
	TG, mmol/L	1.67 a 1.18	1.68 a 1.09	1.73 a 1.34	.39	.94	.21	.26
	LPa, mg/dL	20.47 a 22.19	23.9 a 25.85	25.21 a 25.68				.16
	TB, I'mol/L	13.83 a 6.78	13.42 a 6.22	13.24 a 6.25				.40
	UB, I'mol/L	9.99 a 5.11	9.73 a 4.80	9.53 a 4.83		.08		.20
	CB, I'mol/L	3.84 a 2.62	3.69 a 1.95	3.72 a 2.12	.09		.09	.77
	UA, I'mol/L	348.45 a 105.85	349.86 a 105.61	365.16 a 107.07		.45		
	Cr, I'mol/L	78.82 a 48.23	82.37 a 65.00	86.07 a 56.77		.07		.07
	BUN, mmol/L	5.24 a 1.77	5.35 a 2.22	5.47 a 2.25		.10		.12
	eGFR, mL/min	84.46 a 17.21	83.27 a 18.36	80.49 a 19.91		.06		
Blood routine examinations	WBC, $\times 10^9$	6.39 a 1.77	6.40 a 1.75	6.61 a 1.80		.86		
	Lymphocyte, %	26.74 a 7.98	26.11 a 7.84	25.12 a 7.80				
	Neutrophile, %	62.26 a 10.04	62.87 a 9.89	64.02 a 9.27		.06		
	Plt, $\times 10^9$	179.74 a 53.81	179.22 a 58.43	181.21 a 58.59	.61	.85	.43	.35
	MPV, fL	9.19 a 1.41	9.25 a 1.45	9.12 a 1.37	.05	.24	.19	
	pre-CKMB, IU	14.79 a 10.57	15.55 a 9.11	15.61 a 9.17				.86
	FBG, mg/L	6.30 a 2.19	6.41 a 2.36	6.66 a 2.59		.19		
Medicine	anti-Hyper Med, yes%	77	79	84		.19		
	Statins, yes%	97	98	98	.42	.35	.21	.76
	anti-Plt Med, yes%	100	100	100	.26	.50	.31	.10
Procedure factors	FFR, IVUS, OCT, yes%	12	11	7		.29		
	ACC/AHA TypeB2C, yes%	28	34	38				
	Left coronary artery, yes%	77	79	66				
	Total length of stents, mm	28.12 a 15.07	36.75 a 21.78	47.13 a 24.09				
	Diameter of stent =2.5 mm	91	91	91	.87	.87	.60	.73
	PCI without dilation, yes%	85	87	90		.09		

anti-Hyper Med = anti-hypertension medicine, anti-Plt Med = anti-platelet medicine, BMI = body mass index, BUN = blood urea nitrogen, CB = conjugated bilirubin, CK-MB = creatine kinase MB, Cr = creatinine, DBP = diastolic blood pressure, DM = diabetes mellitus, eGFR = estimated glomerular filtration rate, FBG = fibrinogen, F-CVD = family history of cerebral-or-cardiovascular diseases, FFR = fractional flow reserve, HDL-C = high density lipoprotein cholesterol, Hyper = hypertension, IVUS = intravascular ultrasound, LDL-C = low density lipid cholesterol, LPa = lipid protein alpha, MPV = mean platelet volume, OCT = optical coherence tomography, Plt = platelet, SYB = systolic blood pressure, TB = total bilirubin, TC = total cholesterol, TG = triglyceride, UA = uric acid, UAP = unstable angina pectoris, UB = unconjugated bilirubin, VLDL-C = very low density lipid cholesterol, WBC = white blood cells.

P-value: <.05 means significant statistically.

prescribed 100 mg/d aspirin and 75 mg/d clopidogrel maintenance for more than 7 days before coronary angiography. Two invasive cardiologists who were blinded to the final results performed the coronary angiographies. Three independent cardiologists then calculated final scores for the statistical analysis, following the principles of GS.

2.4. Statistical analysis

All data were collected and analyzed using Statistical Package for the Social Science for Mac OS, version 23 (SPSS inc., Chicago, IL). Categorical variables were presented as a percentage and assessed with the use of chi-square or Fisher's exact test. Continuous variables were presented as mean ± standard deviation and assessed with the use of Student's *t* test or analysis of variance. When comparing differences between groups, the least significant difference post hoc test was selected. Univariate and multivariate logistic regression were then performed to explore the relationship between GS and PMI. The optimal cut-off point of GS was measured via receiver operating characteristic (ROC) curve analysis. A *P*-value of <.05 was considered statistically significant.

3. Results

We recruited 4949 patients with CAD (3366 men, 1583 women; mean age 66.45 ± 10.09 years). Based on the tertile of the GS 20 and 36, the population was divided into 3 groups: Low Group (0 < GS ≤ 20, N = 1809); Intermediate Group (20 < GS ≤ 36, N = 1579); High Group (GS > 36, N = 1561). General information is presented in Table 1.

3.1. Incidence of PMI

The incidence of PMI3 and PMI5 was calculated individually. A direct comparison between different groups is demonstrated in Figure 2. For both PMI3 and PMI5, PMI incidence was

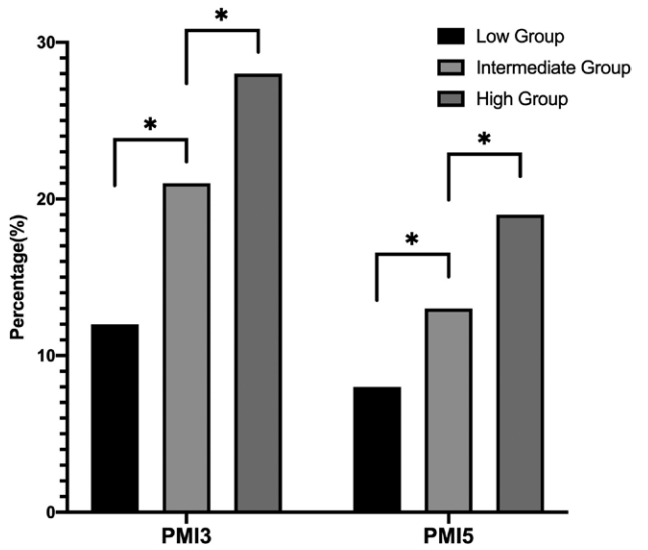

Figure 2. Comparison between different groups for PMI3 and PMI5.

statistically higher in the High Group than in the Intermediate Group, while the Intermediate Group was statistically higher than Low Group.

3.2. Regression analysis between variables and PMI

All the demographic variables and GS were analyzed by univariate logistic regression for PMI3 (see Table 2). Given that the *P*-value of some variables (Gender, Age, Smoking, Hyper, DM, Drinking, F-CVD, HDL-C, LPa, TB, UB, Cr, BUN, eGFR, WBC, Lymphocyte, Neutrophil, pre-CKMB, anti-Hyper Meds, anti-Plt Meds, ACC/AHA type B2C, Left Coronary artery, Total Length of Stents, and GS) was <.10, these attributes were then tested with multivariate logistic regression for PMI3 (Table 2; Odds Ratio in the Fig. 3A). Following the adjustment of these variables, GS remained a statistically significant predictor for PMI3 ($\beta = 0.006$, $P < .05$). Moreover, Age, HDL-C, WBC, pre-CKMB, Left Coronary artery, and Total Length of Stents were also independent predicators for the prediction of PMI3.

Additionally, all the demographic variables and GS were analyzed using univariate logistic regression for PMI5 (see Table 3). Given that the *P*-value of some variables (Gender,

Table 2

Logistic regression results between variables and PMI3.

		Univariate regression		Multivariate regression	
Categories	**Variables**	**ß**	**P**	**ß**	**P**
General information	Gender, male%	–0.174		–0.152	.14
	Age, yr	0.028		0.019	
	BMI, kg/m^2	–0.015	.16	–	–
	SYB, mmHg	0.001	.22	–	–
	DBP, mmHg	0.000	.99	–	–
	UAP, yes%	0.081	.26	–	–
Medical history	Hyper, yes%	0.357		0.160	.09
	DM, yes%	0.248		0.027	.76
	Smoking, yes%	–0.211		–0.113	.26
	Drinking, yes%	–0.268		–0.089	.37
	F-CVD, yes%	–0.257	.05	–0.263	.07
Biochemistry results	TC, mmol/L	–0.302	.31	–	–
	HDL-C, mmol/L	–0.402		–0.378	
	LDL-C, mmol/L	–0.002	.97	–	–
	VLDL-C, mmol/L	0.001	.98	–	–
	TG, mmol/L	–0.042	.19	–	–
	LPa, mg/dL	0.003		0.002	.13
	TB, μmol/L	–0.015		0.024	.18
	UB, μmol/L	0.026		–0.043	.09
	CB, μmol/L	–0.002	.91	–	–
	UA, μmol/L	0.000	.46	–	–
	Cr, μmol/L	0.001		–0.001	.29
	BUN, mmol/L	0.070		0.035	.12
	eGFR, mL/min	–0.014		–0.005	.12
Blood routine examinations	WBC, $\times 10^9$	0.077		0.045	
	Lymphocyte, %	–0.021		–0.008	.27
	Neutrophile, %	0.014		0.000	.94
	Plt, $\times 10^9$	0.000	.49	–	–
	MPV, fL	0.012	.63	–	–
	CK-MB, IU	0.026		0.028	
	FBG, mg/L	0.022	.12	–	–
Medicine	anti-Hyper Med, yes%	0.200		–0.115	.29
	Statins, yes%	–0.171	.45	–	–
	anti-Plt Med, yes%	–0.454	.08	–0.225	.42
Procedure factors	FFR, IVUS, OCT, yes%	0.148	.19	–	–
	ACC/AHA TypeB2C, yes%	0.220		0.018	.83
	Left coronary artery, yes%	–0.191		0.574	
	Total length of stents, mm	0.025		0.024	
	Diameter of stent =2.5 mm	–0.097	.43	–	–
	PCI without dilation, yes%	0.126	.26	–	–
	Gensini score	0.014		0.006	

Abbreviations were the same as Table 1.

P-value: <.05 means significant statistically, attributes with $P < .1$ in univariate results would be selected in multivariate regression.

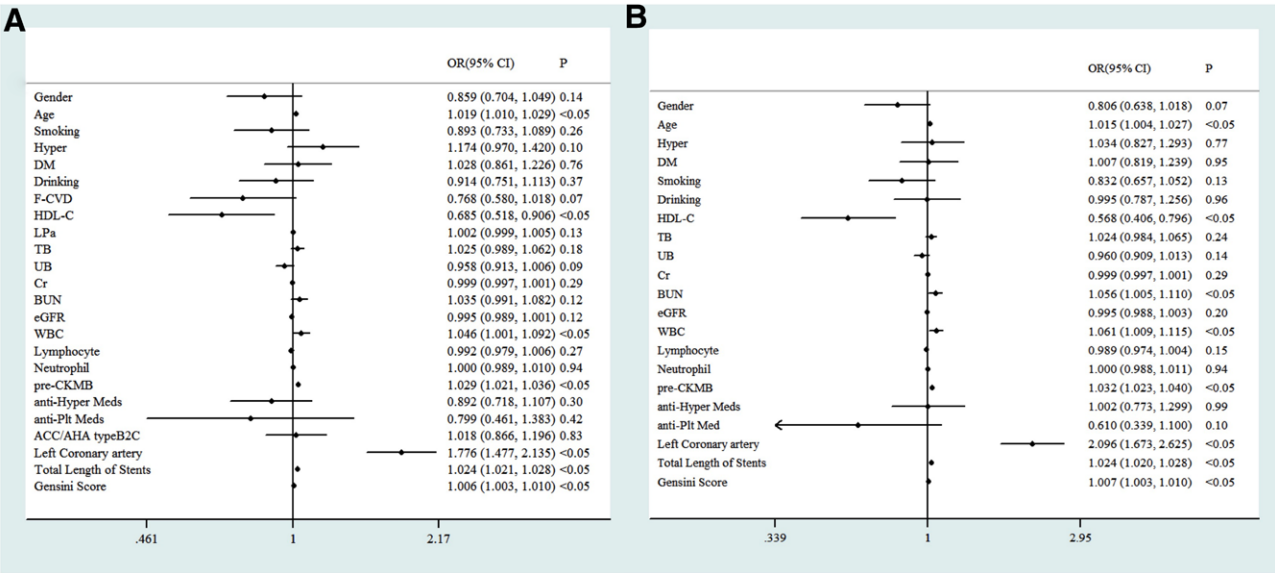

Figure 3. The forest plot of PMI3 (A) and PMI5 (B) for multivariate logistic regression.

Table 3

Logistic regression results between variables and PMI5.

			Univariate regression		Multivariate regression	
Categories	**Variables**		**ß**	**P**	**ß**	**P**
General information	Gender, male%		–0.197		–0.216	.07
	Age, yr		0.024		0.015	
	BMI, kg/m^2		–0.009	.48	–	–
	SYB, mmHg		0.000	.46	–	–
	DBP, mmHg		0.001	.85	–	–
	UAP, yes%		0.108	.21	–	–
Medical history	Hyper, yes%		0.300		0.034	.77
	DM, yes%		0.250		0.007	.95
	Smoking, yes%	T3	–0.251		–0.184	.13
	Drinking, yes%		–0.246		–0.005	.96
	F-CVD, yes%		–0.240	.13	–	–
Biochemistry results	TC, mmol/L		–0.050	.18	–	–
	HDL-C, mmol/L		–0.577		–0.565	
	LDL-C, mmol/L		0.004	.93	–	–
	VLDL-C, mmol/L		–0.039	.32	–	–
	TG, mmol/L		–0.060	.14	–	–
	LPa, mg/dL		0.002	.20	–	–
	TB, μmol/L		–0.014		0.024	.24
	UB, μmol/L		–0.024		–0.041	.14
	CB, μmol/L		0.000	.99	–	–
	UA, μmol/L		0.000	.65	–	–
	Cr, μmol/L		0.001		–0.001	.29
	BUN, mmol/L		0.079		0.054	
	eGFR, mL/min		–0.013		–0.005	.20
Blood routine examinations	WBC, ×10^9		0.100		0.059	
	Lymphocyte, %		–0.024		–0.011	.15
	Neutrophile, %		0.017		0	.94
	Plt, ×10^9		–0.001	.52	–	–
	MPV, fL		0.025	.40	–	–
	CK-MB, IU		0.029		0.031	
	FBG, mg/L		0.027	.11	–	–
Medicine	anti-Hyper Med, yes%		0.251		0.002	.99
	Statins, yes%		–0.165	.53	–	–
	anti-Plt Med, yes%		–0.692		–0.494	.10
Procedure factors	FFR, IVUS, OCT, yes%		0.100	.49	–	–
	ACC/AHA TypeB2C, yes%		0.139	.12	–	–
	Left coronary artery, yes%		0.327		0.740	
	Total length of stents, mm		0.024		0.024	
	Diameter of stent =2.5 mm		–0.096	.51	–	–
	PCI without dilation, yes%		0.124	.36	–	–
	Gensini score		0.014		0.007	

Abbreviations were the same as Table 1.

P-value: <.05 means significant statistically, attributes with $P < .1$ in univariate results would be selected in multivariate regression.

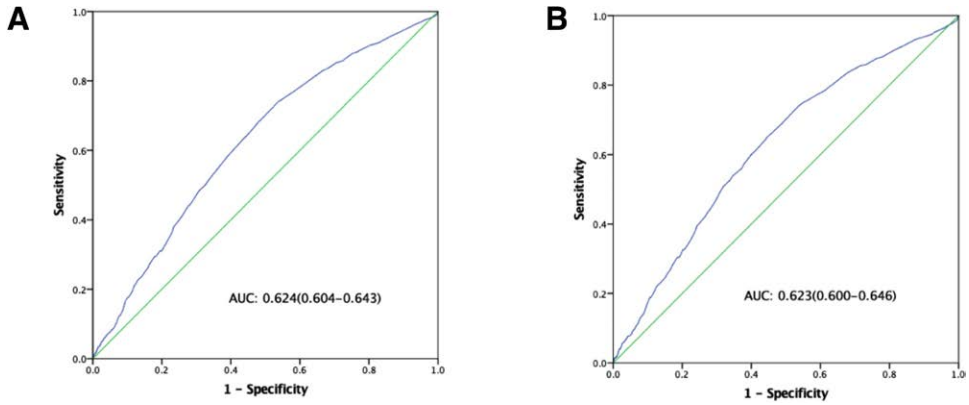

Figure 4. Receiver operating characteristic curve for GS in predicting PMI3 (A) and PMI5 (B).

Table 4

Optimal cut-off value of Gensini score for PMI.

Variables	Optimal cut-off	Sensitivity (%)	Specificity (%)
PMI3	22.5	74.07	46.19
PMI5	27	64.66	55.77

Age, Hyper, DM, Smoking, Drinking, HDL-C, TB, UB, Cr, BUN, eGFR, WBC, Lymphocyte, Neutrophil, pre-CKMB, anti-Hyper Meds, anti-Plt Meds, Left Coronary artery, Total Length of Stents, and GS) was <.10, these attributes were then tested with multivariate logistic regression (see Table 3; Odds Ratio in the Fig. 3B). Following the adjustment of these variables, GS was still an independent significantly predictor for PMI5 ($\beta = 0.007$, $P < .05$). Moreover, Age, HDL-C, BUN, WBC, pre-CKMB, Left Coronary artery, and Total Length of Stents were also independent predicators for the prediction of PMI5.

Furthermore, ROC analysis was used to determine the absolute value of GS for predicting ROC analysis for PMI3 (Fig. 4A) and PMI5 (Fig. 4B). The Youden index was utilized to select the optimal cut-off value for the GS to predict PMI3 (GS = 22.5) and PMI5 (GS = 27) (see Table 4).

4. Discussion

The GS was an independent predictor of PMI following the definitions from the SCAI[13] and the Universal Definition of Myocardial Infarction.[14] Additionally, the 22.5 of GS was the optimal cut-off value for determining the presence of PMI3, while the 27 of GS for PMI5.

Firstly, certain demographic variables were correlated with the presence of PMI in both our study and in previous researches. In this study, regression results indicate that Age, HDL-C, BUN, WBC, pre-CKMB, Left Coronary artery, and Total Length of Stents were independent indicators for predicting PMI. Since 2005, some categories (patient-related risk factors, lesion-related risk factors, procedure-related risk factors) have been known to be related to the presence of PMI.[2] HDL-C was also proved in another article for predicting PMI,[17] as well as VLDL-C and LPa in diabetic patients,[18] but not in our study. About inflammation with PMI, a higher Neutrophil-to-Lymphocyte ratio increases the risk of PMI from an original article,[19] also WBC may show a power for predicting in consistent with our results. Besides procedural-related risk factors in 2005, Left Coronary artery, and Total Length of Stents were also showed their predicting power for PMI in an Korean research[20] and in our results. However, other variables, such as CRP,[21] LDL-C,[2] and TC,[14] weren't found to be related with PMI in our study.

Secondly, GS was a significant indicator for PMI presence. Many different scores calculated the severity of CAD in coronary angiography, such as SYNTAX score,[5] ACC/AHA score,[6] LEAMAN score,[7] and GS. In a 2013 study,[20] Kenneth initially defined the relationship between SYNTAX score and PMI, and concluded that the SYNTAX score was able to stratify PMI risk. Like SYNTAX score, GS was another convenient and powerful severity score for determining CAD. Many authors have reported its function in various cardiological fields. For example, Sayin has reported that the value of GS represented the degree of CAD correlated with Framingham risk score in the Turkey population.[22] Additionally, Zhenhong[23] group reported that GS was an effective parameter for predicting long-term mortality in acute coronary syndrome patients. In another 2016 study on multivessel therapy and the risk of PMI, Zhangwei[9] also demonstrated the relationship between GS and PMI in the results, although specific details weren't shown in that article. In our study, the relationship between GS and PMI was again proven, while the power of GS was determined by ROC analysis, with an optimal cut-off value of 22.5 for PMI3 and 27 for PMI5. The preprocedural assessment of the GS could help identify patients with a high risk of PMI, based on the optimal values showing above.

Additionally, various definitions have been put forward in the past few decades. The fourth universal definition of myocardial infarction had been established by the European Society of Cardiology, the American College of Cardiology, the American Heart Association and the World Health Organization in 2019.[14] In these guidelines, PCI-related myocardial infarction was defined as a cTnI > 5-fold URL associated with an assisted test result change within 48 hours. While this definition was the latest, not many authors have supported the validation of the 5-fold cut-off value. Given this, our study extended the results of universal definition as 3-fold and 5-fold to explore the potential relationship between GS and PMI.

For the management strategies for PMI, some guidelines provided 2 aspects to deal with the patients, including prevention and management. About the prevention, almost all strategies could be divided into 4 groups, such as Antiplatelet therapy (aspirin, adequate clopidogrel preloading, glycoprotein IIb/IIIa antagonists if ACS or complicated PCI),[24] Statin therapy initiated before PCI, Embolic protection in saphenous venous graft intervention, and Ischemic preconditioning. About the management, we must take an intensive secondary prevention, such as LDL goal <70 mg/dL (similar to spontaneous myocardial infarction).[25] Moreover, Post-PCI angina, serum biomarkers elevating (CK-MB, cTnI, etc) associated with ischemic ECG changes may dictate further interventional procedures depending on the amount of myocardium at risk.[13] Generally speaking, we should evaluate the risk of myocardial infarction along with the undergoing process, before PCI, during PCI, and after PCI, earlier diagnosis means the safer treatment for the patients.

5. Limitations

This research has several limitations. Firstly, this was a single-center, retrospective study with patients who were admitted for CAD which excluded healthy people. Secondly, preprocedural related factors weren't included in the statistical analysis due to loss of the operation record in the system upgrading process. Thirdly, in addition to PMI, major adverse cardiac events might be considered, such as all-cause death, fatal or nonfatal myocardial infarction, repeat PCI or bypass surgery in a long-time period. Finally, subgroup analysis could be also analyzed in the future studies, such as DM or smoking status.

6. Conclusions

The GS was an independent predictor of PMI in the single-coronary revascularization population. Additionally, the 22.5 of GS was the optimal cut-off value for determining the presence of PMI3, while the 27 of GS for PMI5.

Author contributions

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